history of one twin. This migration is interpreted to be a reinfection event (a phenomenon which has previously been difficult to identify and quantify in CMV infections). We are currently examining migrant tracts from the ghost population and calculating the timing of the reinfection event.

Conclusion: This study provides a highly detailed model of the population dynamics of CMV in congenital infections and is an important case study of the applicability of population genetic inference modeling to illuminate medically important parameters of viral infections.

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Time: 12:45-14:15
Room: Poster & Exhibition Area

West Nile meningoencephalitis in children

G. Kuli-Lito[1,*], L. Rosa[2], E. Kallfa[3], F. Zavalani[3], H. Hoxha[4], R. Petrela[5], A. Kallfa[6], S. Bino[7]

[1] *University Hospital Center "Mother Teresa", Tirana, Albania*
[2] *University Hospital Center, Tirana, Albania*
[3] *University Hospital Centre "Mother Theresa", Tirana, AL, Albania*
[4] *University hospital center "Mother Theresa", Tirana, Albania, Albania*
[5] *University Hospital Center Mother Theresa Tirana, Albania, Tirana, Albania*
[6] *QSUT, Tirana, Albania, Albania*
[7] *Institute of Public Health, Tirane, Albania*

Background: West Nile virus was one of the most frequent causative agents of viral meningoencephalitis in Albania last 3 years. This increased incidence is documented even in pediatric meningoencephalitis occurred during summer period.

Objectives: To study the epidemiological and clinical characteristics and eventual complications in children with confirmed infection.

Methods: The hospitalized children, who fulfill the diagnostic criteria for West Nile meningoencephalitis, during the last three years, were included at this report. The epidemiologic data analyzed were: age, gender, locality, seasonal distribution, time of hospitalization. The confirmation of infection was performed by specific serologic examination of paired serum samples of children diagnosed with meningoencephalitis.

Results: 7 children with confirmed serological diagnosis were prescribed at the study. The age range was 4y-13 years. The dominating case were boys coming from urban areas. The greatest number of cases was presented during the month of August, followed by September and July. The clinical picture was dominated by high fever, vomiting and the presence of meningeal syndrome. A moderate mononuclear lcs pleocytosis, ranged from 25-400 cells/ml, was observed in all children. The average of fever period was 4 day, while clinical signs persisted longer with an average of 7 days. The recovery was complete with no complications after a course with symtomatic treatment.

Conclusion: The meningoencephalitis is an important infection in children. The virus of West Nile should be considered as the possible causative agent as long as the transmitter vector; mosquitoes are spread all around the world especially in Mediterranean region.

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Genetic analysis of the complete NS gene of novel pandemic influenza A H1N1 2009 virus strains circulating in India during 2010-11

S. Kumar[1,*], I. Gadhoke[1], S. khare[2], D.S. Rawat[3], L.S. Chauhan[4], A. Rai[2]

[1] *National Centre for Disease Control, Delhi, Delhi, India*
[2] *National Centre for Disease Control, Delhi, India*
[3] *National Centre for Disease Control, 110054, Delhi, India*
[4] *National Centre for Disease Control, Delhi, New Delhi, India*

Background: Influenza is responsible for epidemics and pandemics across the globe. NS1 protein encoded by the co-linear mRNA derived from segment 8 of the influenza A virus. NS1 protein consists of two functional domains *i)*N-terminal RNA binding domain (RBD) (residues 1-72) involved in sequestering of dsRNA and preventing the activation of antiviral enzyme PKR/2'-5' oligo(A) synthetase. *ii)* C-terminal Effector domain, ED (residues 84-220) mediates interaction with host cell proteins. It acts as interferon antagonist. The present study was carried out to study emerging mutations in RNA binding and effector domain (ED) of NS1 gene of influenza A H1N1 2009.

Methods: Eighty nasal and throat swab samples in VTM were collected at NCDC from north Indian region and stored at -800c. Samples diagnosed for the presence of Influenza A virus by TaqMan Real time PCR for PdmH1N1 2009 panel. Full-length gene sequencing 16 positive samples was performed for segment 8 of pdm H1N1 2009 by M13-tailed sequencing primers for NS gene by dideoxy chain termination method and sequenced on ABI 3130xl Genetic Analyser. Sequence analysis was done using Bio-Edit version 7.09 and phylogenetic analysis was done using the MEGA version 5.

Results: All the 16 samples had CT value below 32 for all four targets of pdm H1N1 2009 panel. Compared to reference strain FJ969538.1as, three significant (E51Q, D53N & S73T) were observed in the RNA binding domain (RBD); and five mutations Mutations (E96K, R108K, T143N, I145V and N209D) were found in the Effector domain (ED) of NS1 gene. Single mutation (I123V) was conserved in the ED domain of all the 16 samples.

Conclusion: Sequencing data analysis shows rapidly mutating ED compared to RBD in NS1 gene; which may affect interferon production. None of the mutations fell in the conserved basic residues involved in interaction with dsRNA. E96K mutation may result in lack of inhibition of Interferon induction by circulating virus strain. No changes were seen in NS2 (NEP). NS1 is an interferon antagonist and any mutation in NS1 may affect virulence and effective virus replication in host cell.

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